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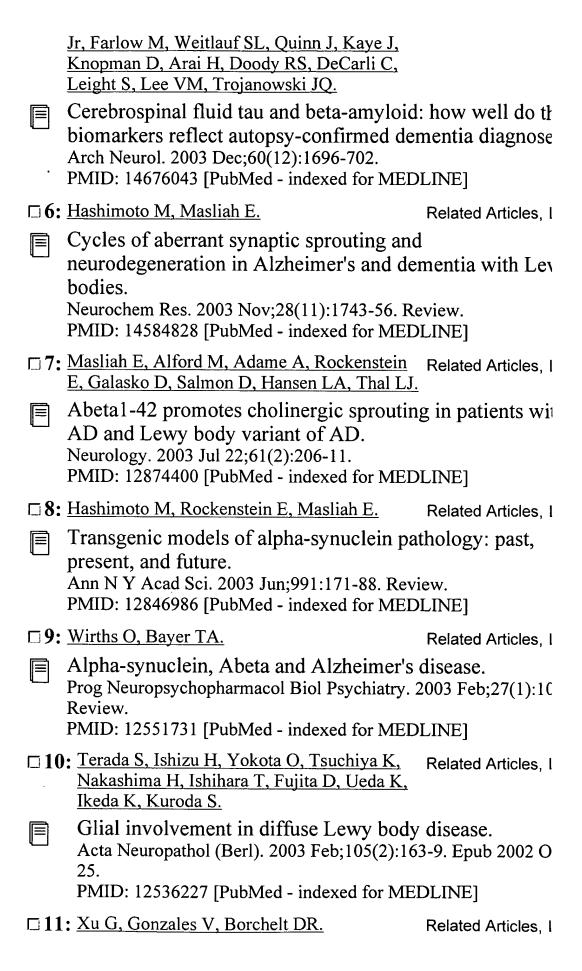


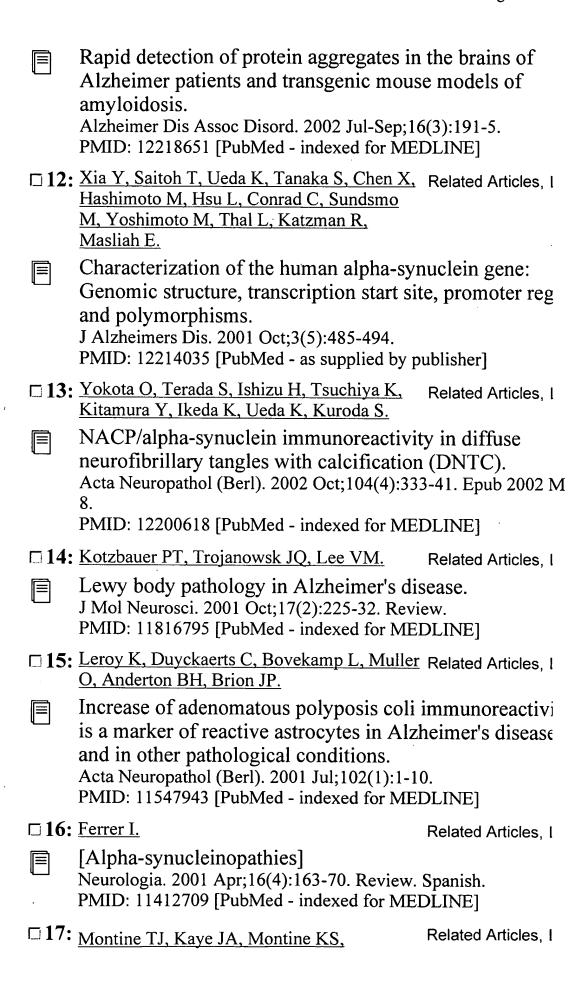


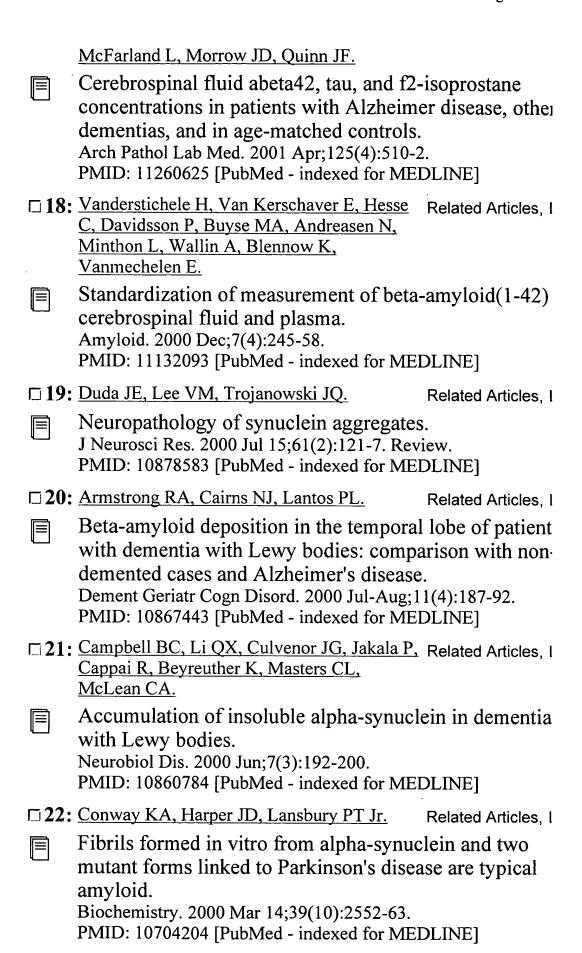


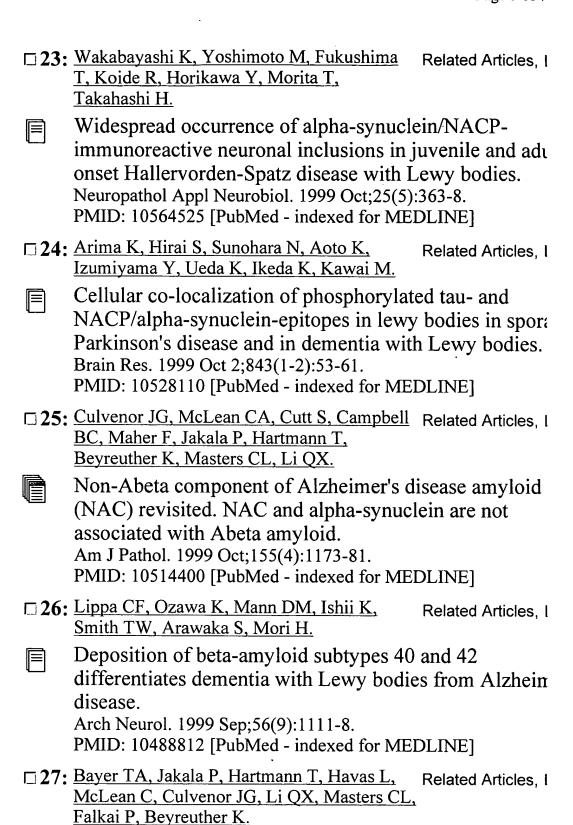


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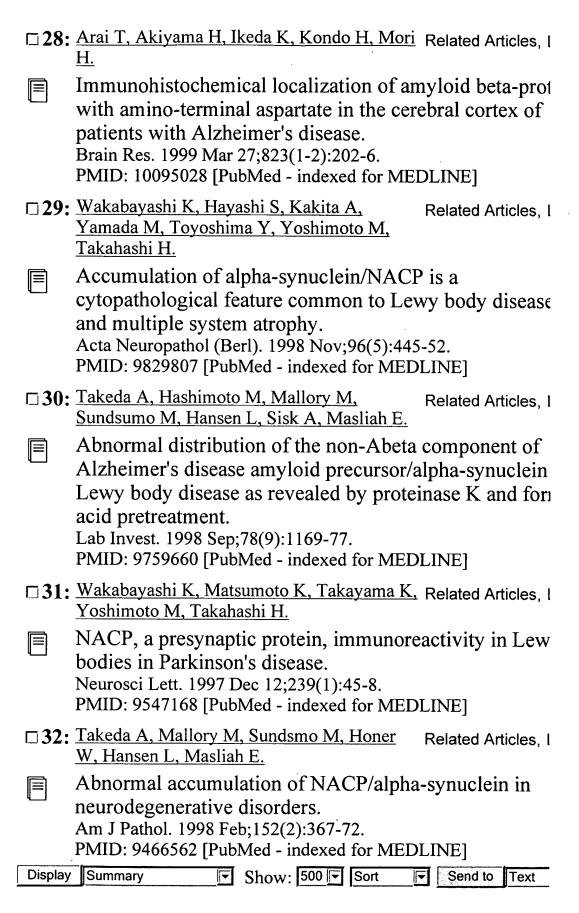








Alpha-synuclein accumulates in Lewy bodies in Parkinson's disease and dementia with Lewy bodies but in Alzheimer's disease beta-amyloid plaque cores. Neurosci Lett. 1999 May 14;266(3):213-6. PMID: 10465711 [PubMed - indexed for MEDLINE]



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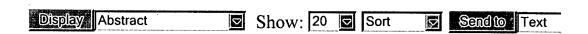
Abnormal accumulation of NACP/alpha-synucle in neurodegenerative disorders.

Takeda A, Mallory M, Sundsmo M, Honer W, Hansen Masliah E.

Department of Neurosciences, University of California, S Diego, School of Medicine La Jolla, 92093-0624, USA.

The precursor of the non-Abeta component of Alzheimer' disease amyloid (NACP) (also known as a-synuclein) is a presynaptic terminal molecule that accumulates in the pla of Alzheimer's disease. Recent studies have shown that a mutation in NACP is associated with familial Parkinson's disease, and that Lewy bodies are immunoreactive with antibodies against this molecule. To clarify the patterns of accumulation and differences in abnormal compartmentalization, we studied NACP immunoreactivity using double immunolabeling and laser scanning confoca microscopy in the cortex of patients with various neurodegenerative disorders. In Lewy body variant of Alzheimer's disease, diffuse Lewy body disease, and Parkinson's disease, NACP was found to immunolabel cortical Lewy bodies, abnormal neurites, and dystrophic neurites in the plaques. Double-labeling studies showed th all three of these neuropathological structures also contain ubiquitin, synaptophysin, and neurofilament (but not tau) immunoreactivity. In contrast, neurofibrillary tangles, neuropil threads, Pick bodies, ballooned neurons, and glia tangles (most of which were tau positive) were NACP negative. These results support the view that NACP specifically accumulates in diseases related to Lewy bodic such as Lewy body variant of Alzheimer's disease, diffuse Lewy body disease, and Parkinson's disease and suggests role for this synaptic protein in the pathogenesis of neurodegeneration.

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NACP, a presynaptic protein, immunoreactivity Lewy bodies in Parkinson's disease.

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Brain Disease Research Center, Niigata University, Japan koichi@bri.niigata-u.ac.jp

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NACP, originally identified as a precursor of the non-Abe component of Alzheimer's disease amyloid (NAC), is nov known to be identical to alpha-synuclein, a presynaptic protein in the human brain. Recently, a mutation in the all synuclein gene in families with autosomal dominant Parkinson's disease (PD) was identified. We carried out immunohistochemical examinations of the brains of spora PD patients using anti-NACP and anti-ubiquitin antibodie Consistent with previous studies, the anti-NACP antibody immunostained the neuropil in a punctate pattern through the brain. Moreover, much stronger NACP immunoreactive was found in Lewy bodies and degenerating neurites in th brainstem. Serial sections immunolabeled with anti-ubiqu or anti-NACP showed that all ubiquitin-immunoreactive I were also NACP-immunoreactive. These findings suggest that alteration of NACP metabolism is involved in the pathogenesis of PD, particularly in Lewy body formation.

leading to neurodegeneration.

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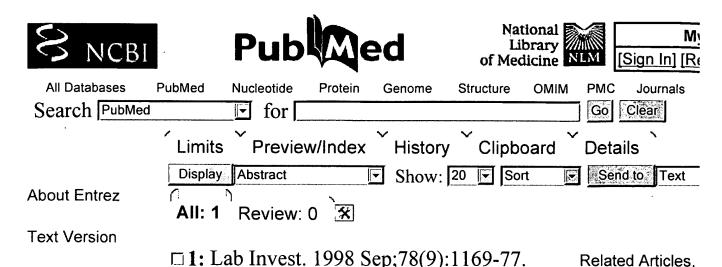
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Abnormal distribution of the non-Abeta compor of Alzheimer's disease amyloid precursor/alphasynuclein in Lewy body disease as revealed by proteinase K and formic acid pretreatment.

Takeda A, Hashimoto M, Mallory M, Sundsumo M, Hansen L, Sisk A, Masliah E.

Department of Neurosciences, University of California, S Diego, School of Medicine, La Jolla 92093-0624, USA.

The precursor of the non-Abeta component of Alzheimer' disease amyloid (NACP) (also known as alpha-synuclein) presynaptic terminal molecule that abnormally accumulat the plaques of Alzheimer's disease (AD) and in the Lewy bodies (LBs) of Lewy body variant of AD, diffuse Lewy body disease, and Parkinson's disease. To better understar the distribution of NACP/alpha-synuclein and its fragmen the LB-bearing neurons and neurites, as well as to clarify patterns of NACP/alpha-synuclein compartmentalization, studied NACP/alpha-synuclein immunoreactivity using antibodies against the C-terminal, N-terminal, and NAC regions after Proteinase K and formic acid treatment in the cortex of patients with LBs. Furthermore, studies of the subcellular localization of NACP/alpha-synuclein within bearing neurons were performed by immunogold electron

microscopy. These studies showed that the N-terminal antibody immunolabeled the LBs and dystrophic neurites with great intensity and, to a lesser extent, the synapses. In contrast, the C-terminal antibody strongly labeled the synapses and, to a lesser extent, the LBs and dystrophic neurites. Whereas Proteinase K treatment enhanced NACP/alpha-synuclein immunoreactivity with the C-term antibody, it diminished the N-terminal NACP/alpha-synuc immunoreactivity. Furthermore, formic acid enhanced LE and dystrophic neurite labeling with both the C- and Nterminal antibodies. In addition, whereas without pretreatment only slight anti-NAC immunoreactivity was found in the LBs, formic acid pretreatment revealed an extensive anti-NAC immunostaining of LBs, plaques, and glial cells. Ultrastructural analysis revealed that NACP/all synuclein immunoreactivity was diffusely distributed with the amorphous electrodense material in the LBs and as sm clusters in the filaments of LBs and neurites. These result support the view that aggregated NACP/alpha-synuclein might play an important role in the pathogenesis of disord associated with LBs.

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Accumulation of alpha-synuclein/NACP is a cytopathological feature common to Lewy body disease and multiple system atrophy.

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Wakabayashi K, Hayashi S, Kakita A, Yamada M, Toyoshima Y, Yoshimoto M, Takahashi H.

(Cubby)

Brain Disease Research Center, Brain Research Institute, Niigata University, Japan. koichi@bri.niigata-u.ac.jp

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Recently, we have shown that the precursor of the non-Al component of Alzheimer's disease amyloid (NACP), also known as alpha-synuclein, is a major component of Lewy bodies (LBs) as well as neuronal and glial cytoplasmic inclusions in multiple system atrophy (MSA). To elucidat whether the accumulation of NACP is specific to LB dise. and MSA, we further studied 83 autopsied cases with vari neurological disorders, using anti-NACP antibodies. In Ll disease, NACP immunoreactivity was present in all of the LBs and Lewy neurites in both the central and peripheral nervous systems, the pale bodies in the substantia nigra, a dystrophic neurites in the hippocampal CA2/3 region. Immunoelectron microscopy revealed that the reaction product was localized within filamentous structures and associated granular structures. In MSA, NACP

immunoreactivity was found in the intracytoplasmic inclusions of both neuronal and oligodendroglial cells, neuronal intranuclear inclusions, and swollen neuronal processes. No NACP immunoreactivity was found in a variety of other neuronal or glial inclusions in other disordincluding Alzheimer's disease, Pick's disease, progressive supranuclear palsy, corticobasal degeneration, motor neur disease and triplet-repeat diseases. These findings strongly suggest that the accumulation of NACP is a cytopathologic feature common to LB disease and MSA.

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cytopathological feature common to Lewy body disease and multiple system atrophy.

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Wakabayashi K, Hayashi S, Kakita A, Yamada M, Toyoshima Y, Yoshimoto M, Takahashi H.

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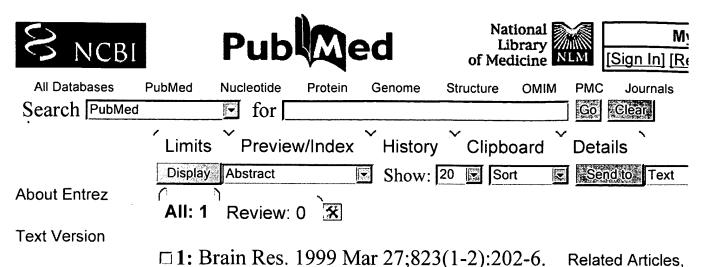
Recently, we have shown that the precursor of the non-Al component of Alzheimer's disease amyloid (NACP), also known as alpha-synuclein, is a major component of Lewy bodies (LBs) as well as neuronal and glial cytoplasmic inclusions in multiple system atrophy (MSA). To elucidat whether the accumulation of NACP is specific to LB dise and MSA, we further studied 83 autopsied cases with vari neurological disorders, using anti-NACP antibodies. In Ll disease, NACP immunoreactivity was present in all of the LBs and Lewy neurites in both the central and peripheral nervous systems, the pale bodies in the substantia nigra, a dystrophic neurites in the hippocampal CA2/3 region. Immunoelectron microscopy revealed that the reaction product was localized within filamentous structures and associated granular structures. In MSA, NACP

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Immunohistochemical localization of amyloid be protein with amino-terminal aspartate in the cerebral cortex of patients with Alzheimer's disease.

Arai T, Akiyama H, Ikeda K, Kondo H, Mori H.

Department of Neuropathology, Tokyo Institute of Psychiatry, 2-1-8 Kamikitazawa, Setagaya-ku, Tokyo 156 8585, Japan. arai@prit.go.jp

We investigated immunohistochemically the localization amyloid beta-protein (Abeta) with amino-terminal asparta (N1[D]) in brains of patients with Alzheimer's disease, diffuse Lewy body disease and Down's syndrome. A monoclonal antibody, 4G8, which recognizes the middle portion of Abeta, was used as a reference antibody to labe the total Abeta deposits. Double staining with anti-Abeta([D]) and 4G8 revealed that Abeta deposits in the subiculu and the neocortical deep layers often lacked N1[D] immunoreactivity, indicating N-terminal truncation of Ab in these deposits. Abeta deposits in the neocortical superf layers and the presubicular parvopyramidal layer always contained Abeta with N1[D]. Such regional as well as lam differences in the distribution of Abeta beginning at N1[D suggest that some local factors influence N-terminal

processing of Abeta deposited in the brain. Copyright 199 Elsevier Science B.V.

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Alpha-synuclein accumulates in Lewy bodies in Parkinson's disease and dementia with Lewy bot but not in Alzheimer's disease beta-amyloid plac cores.

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Bayer TA, Jakala P, Hartmann T, Havas L, McLean C Culvenor JG, Li QX, Masters CL, Falkai P, Beyreuthe K.

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Department of Psychiatry, University of Bonn Medical Center, Germany. bayer@uni-bonn.de

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A growing body of evidence suggests that the non-Abeta component of Alzheimer's disease amyloid precursor prot (NACP) or alpha-synuclein contributes to the neurodegenerative processes in Alzheimer's disease (AD) Parkinson's disease (PD) and dementia with Lewy bodies (DLB). In the present study antisera to the N terminus and NAC domain of the alpha-synuclein protein were employto elucidate the expression pattern in brains of patients wi AD, PD, DLB and control specimen. Alpha-synuclein exhibited an overall punctuate expression profile compati with a synaptic function. Interestingly, while Lewy bodies were strongly immunoreactive, none of the alpha-synucle antisera revealed staining in mature beta-amyloid plaques AD. These observations suggest that alpha-synuclein does

contribute to late neurodegenerative processes in AD brai

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ARCH NEUROL Deposition of beta-amyloid subtypes 40 and 42 differentiates dementia with Lewy bodies from Alzheimer disease.

Lippa CF, Ozawa K, Mann DM, Ishii K, Smith TW, Arawaka S, Mori H.

Department of Neurology, MCP-Hahnemann University, Philadelphia, PA 19129, USA. lippa@auhs.edu

BACKGROUND: Alterations in the metabolism of the amyloid precursor protein and the formation of beta-amyl (Abeta) plagues are associated with neuronal death in Alzheimer disease (AD). The plaque subtype Abeta(x-42)occurs as an early event, with Abeta(x-40) plaques formir a later stage. In dementia with Lewy bodies (DLB), an increase in the amount of cortical Abeta occurs without severe cortical neuronal losses. OBJECTIVE: To advance understanding of the natural history of Abeta in neurodegenerative diseases. DESIGN: We evaluated the expression of Abeta(x-40) and Abeta(x-42) in DLB using monoclonal antibodies and immunohistochemical techniq in 5 brain regions. The data were compared with those elicited with normal aging and from patients with AD. SETTING AND PATIENTS: A postmortem study involv 19 patients with DLB without concurrent neuritic

degeneration, 10 patients with AD, and 17 aged persons without dementia for control subjects. RESULTS: The Al plaques were more numerous in patients with DLB than it controls in most brain regions, although the Abeta(x-42) plaque subtype was predominant in both conditions. Over Abeta(x-42) plague density was similar in patients with D and those with AD, but Abeta(x-40) plaques were more numerous in persons with AD than in those with DLB. Th ratio of Abeta(x-40) to Abeta(x-42) plaques was significa reduced in persons with DLB compared with patients witl AD. CONCLUSIONS: The Abeta plaques were more numerous in patients with DLB than persons with normal aging, but the plaque subtypes were similar. The relative proportion of the 2 Abeta plaque subtypes in DLB is distinguishable from that in AD.

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Deposition of beta-amyloid subtypes 40 and 42 differentiates dementia with Lewy bodies from Alzheimer disease.

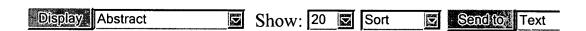
Lippa CF, Ozawa K, Mann DM, Ishii K, Smith TW, Arawaka S, Mori H.

Department of Neurology, MCP-Hahnemann University, Philadelphia, PA 19129, USA. lippa@auhs.edu

BACKGROUND: Alterations in the metabolism of the amyloid precursor protein and the formation of beta-amyl (Abeta) plagues are associated with neuronal death in Alzheimer disease (AD). The plaque subtype Abeta(x-42) occurs as an early event, with Abeta(x-40) plaques formir a later stage. In dementia with Lewy bodies (DLB), an increase in the amount of cortical Abeta occurs without severe cortical neuronal losses. OBJECTIVE: To advance understanding of the natural history of Abeta in neurodegenerative diseases. DESIGN: We evaluated the expression of Abeta(x-40) and Abeta(x-42) in DLB using monoclonal antibodies and immunohistochemical techniq in 5 brain regions. The data were compared with those elicited with normal aging and from patients with AD. SETTING AND PATIENTS: A postmortem study involv 19 patients with DLB without concurrent neuritic

degeneration, 10 patients with AD, and 17 aged persons without dementia for control subjects. RESULTS: The At plaques were more numerous in patients with DLB than it controls in most brain regions, although the Abeta(x-42) plaque subtype was predominant in both conditions. Over Abeta(x-42) plaque density was similar in patients with D and those with AD, but Abeta(x-40) plaques were more numerous in persons with AD than in those with DLB. Th ratio of Abeta(x-40) to Abeta(x-42) plaques was significated reduced in persons with DLB compared with patients with AD. CONCLUSIONS: The Abeta plaques were more numerous in patients with DLB than persons with normal aging, but the plaque subtypes were similar. The relative proportion of the 2 Abeta plaque subtypes in DLB is distinguishable from that in AD.

PMID: 10488812 [PubMed - indexed for MEDLINE]



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• Am J Pathol. 2000 Feb;156(2):734-6.

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Non-Abeta component of Alzheimer's disease amyloid (NAC) revisited. NAC and alpha-synuclare not associated with Abeta amyloid.

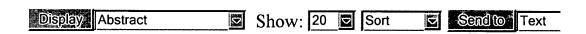
Culvenor JG, McLean CA, Cutt S, Campbell BC, Mał F, Jakala P, Hartmann T, Beyreuther K, Masters CL, QX.

Department of Pathology, The University of Melbourne, Parkville, Victoria, Australia.

alpha-Synuclein (alphaSN), also termed the precursor of t non-Abeta component of Alzheimer's disease (AD) amyle (NACP), is a major component of Lewy bodies and Lewy neurites pathognomonic of Parkinson's disease (PD) and dementia with Lewy bodies (DLB). A fragment of alphaS termed the non-Abeta component of AD amyloid (NAC) previously been identified as a constituent of AD amyloid plaques. To clarify the relationship of NAC and alphaSN Abeta plaques, antibodies were raised to three domains of alphaSN. All antibodies produced punctate labeling of hur cortex and strong labeling of Lewy bodies. Using antibod

to alphaSN(75-91) to label cortical and hippocampal secti of pathologically proven AD cases, we found no evidence NAC in Abeta amyloid plaques. Double labeling of tissue sections in mixed DLB/AD cases revealed alphaSN in dystrophic neuritic processes, some of which were in clos association with Abeta plagues restricted to the CA1 hippocampal region. In brain homogenates alphaSN was predominantly recovered in the cytosolic fraction as a 16protein on Western analysis; however, significant amount aggregated and alphaSN fragments were also found in ure extracts of SDS-insoluble material from DLB and PD case NAC antibodies identified an endogenous fragment of 6 k the cytosolic and urea-soluble brain fractions. This fragma may be produced as a consequence of alphaSN aggregatic alternatively may accelerate aggregation of the full-length alphaSN.

PMID: 10514400 [PubMed - indexed for MEDLINE]



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Non-Abeta component of Alzheimer's disease amyloid (NAC) revisited. NAC and alpha-synuclare not associated with Abeta amyloid.

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PMID: 10514400 [PubMed - indexed for MEDLINE]

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Cellular co-localization of phosphorylated tau- a NACP/alpha-synuclein-epitopes in lewy bodies in sporadic Parkinson's disease and in dementia wi Lewy bodies.

Arima K, Hirai S, Sunohara N, Aoto K, Izumiyama Y. Ueda K, Ikeda K, Kawai M.

Department of Ultrastructure and Histochemistry, Tokyo Institute of Psychiatry, 2-1-8 Kamikitazawa, Setagaya-ku. Tokyo, Japan. arima@prit.go.jp

The precursor of the non-Abeta-component of Alzheimer' disease (AD) amyloid (NACP, alpha-synuclein) aggregate into insoluble filaments of Lewy bodies (LBs) in Parkinso disease (PD) and dementia with LBs (DLB). The microtubule-associated protein tau is an integral compone of filaments of neurofibrillary tangles (NFTs). NFTs are occasionally found in brains of PD and DLB; however, th presence of NFTs or tau-epitopes within LB-containing neurons is rare. Double-immunofluorescence study and peroxidase-immunohistochemical study in serial sections. performed to examine the co-localization of tau- and NAC epitopes in the brainstem of PD and DLB, demonstrated t four different epitopes of tau including phosphorylationdependent and independent ones were present in a minori

LBs, but more often than previously considered. A tau (ta epitope was localized to filaments in the outer layers of brainstem-type LBs by immunoelectron microscopy. Therefore, we conclude that tau is incorporated into filam in certain LBs. Extensive investigation has enabled us to classify this co-localization into four types: type 1, LBs w ring-shaped tau-immunoreactivity; type 2, LBs surrounde NFTs; type 3, NACP- and tau-immunoreactive filamentou and granular masses; and type 4, NACP- and tau-immunoreactive dystrophic neurites. This study raises a n question whether aggregation and hyperphosphorylation of tau in PD and DLB are triggered by the collapse of intraneuronal organization of microtubules due to NACP-filament aggregation in neuronal perikarya and axons.

PMID: 10528110 [PubMed - indexed for MEDLINE]

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NACP/alpha-synuclein-epitopes in lewy bodies in sporadic Parkinson's disease and in dementia wi Lewy bodies.

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Arima K, Hirai S, Sunohara N, Aoto K, Izumiyama Y, Ueda K, Ikeda K, Kawai M.

Department of Ultrastructure and Histochemistry, Tokyo Institute of Psychiatry, 2-1-8 Kamikitazawa, Setagaya-ku. Tokyo, Japan. arima@prit.go.jp

The precursor of the non-Abeta-component of Alzheimer'

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The precursor of the non-Abeta-component of Alzheimer' disease (AD) amyloid (NACP, alpha-synuclein) aggregate into insoluble filaments of Lewy bodies (LBs) in Parkinso disease (PD) and dementia with LBs (DLB). The microtubule-associated protein tau is an integral compone of filaments of neurofibrillary tangles (NFTs). NFTs are occasionally found in brains of PD and DLB; however, the presence of NFTs or tau-epitopes within LB-containing neurons is rare. Double-immunofluorescence study and peroxidase-immunohistochemical study in serial sections, performed to examine the co-localization of tau- and NAC epitopes in the brainstem of PD and DLB, demonstrated to the four different epitopes of tau including phosphorylation-dependent and independent ones were present in a minorical study in a minorical study in a minorical study in serial sections, performed to examine the co-localization of tau- and NAC epitopes in the brainstem of PD and DLB, demonstrated to the study of the present in a minorical study in a minorical study in serial sections, performed to examine the co-localization of tau- and NAC epitopes in the brainstem of PD and DLB, demonstrated to the present in a minorical study in serial sections, performed to examine the co-localization of tau- and NAC epitopes in the brainstem of PD and DLB, demonstrated to the present in a minorical study in serial section of tau- and NAC epitopes in the brainstem of PD and DLB, demonstrated to the present epitopes of tau including phosphorylation-

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PMID: 10528110 [PubMed - indexed for MEDLINE]

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Widespread occurrence of alpha-synuclein/NAC immunoreactive neuronal inclusions in juvenile adult-onset Hallervorden-Spatz disease with Lev bodies.

Wakabayashi K, Yoshimoto M, Fukushima T, Koide I Horikawa Y, Morita T, Takahashi H.

Brain Disease Research Center, Brain Research Institute, Niigata University, Japan.

Alpha-Synuclein (originally called precursor of the non-Abeta component of Alzheimer's disease amyloid-NACP) presynaptic nerve terminal protein and is now known to b major component of Lewy bodies (LBs) in Parkinson's disease. Previous studies have shown that LBs are occasionally found in patients with Hallervorden-Spatz disease (HSD), a hereditary or sporadic neuroaxonal dystrophy. Therefore, an immunocytochemical examination of the brain tissues from two patients with HSD for alpha synuclein/NACP was performed. In both cases, LBs were observed in the substantia nigra, locus ceruleus and other subcortical nuclei. These LBs were strongly immunolabel with anti-alpha-synuclein/NACP. Moreover, abnormal alp

synuclein/NACP-immunoreactive structures in the neuror somata and processes were found in the cerebral neocorte hippocampus, basal ganglia, thalamus, pontine and inferic olivary nuclei, spinal grey matter, and peripheral sympath ganglia. Although numerous dystrophic axons (spheroids) were found throughout the brain, either none or only a few were positive for alpha-synuclein/NACP. These findings suggest that widespread accumulation of alpha-synuclein/NACP is a pathological feature in patients suffe from HSD with LBs, and that this phenomenon is unrelate axonal spheroid formation.

PMID: 10564525 [PubMed - indexed for MEDLINE]

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Fibrils formed in vitro from alpha-synuclein and two mutant forms linked to Parkinson's disease typical amyloid.

Conway KA, Harper JD, Lansbury PT Jr.

Center for Neurologic Diseases, Brigham and Women's Hospital and Department of Neurology, Harvard Medical School, Boston, Massachusetts 02115, USA.

Two missense mutations in the gene encoding alphasynuclein have been linked to rare, early-onset forms of Parkinson's disease (PD). These forms of PD, as well as tl common idiopathic form, are characterized by the present cytoplasmic neuronal deposits, called Lewy bodies, in the affected region of the brain. Lewy bodies contain alphasynuclein in a form that resembles fibrillar Abeta derived from Alzheimer's disease (AD) amyloid plaques. One of t mutant forms of alpha-synuclein (A53T) fibrillizes more rapidly in vitro than does the wild-type protein, suggesting that a correlation may exist between the rate of in vitro fibrillization and/or oligomerization and the progression c PD, analogous to the relationship between Abeta fibrilliza in vitro and familial AD. In this paper, fibrils generated in

vitro from alpha-synuclein, wild-type and both mutant for are shown to possess very similar features that are characteristic of amyloid fibrils, including a wound and predominantly unbranched morphology (demonstrated by atomic force and electron microscopies), distinctive dyebinding properties (Congo red and thioflavin T), and antiparallel beta-sheet structure (Fourier transform infrare spectroscopy and circular dichroism spectroscopy). alpha-Synuclein fibrils are relatively resistant to proteolysis, a property shared by fibrillar Abeta and the disease-associat fibrillar form of the prion protein. These data suggest that like AD, is a brain amyloid disease that, unlike AD, is characterized by cytoplasmic amyloid (Lewy bodies). In addition to amyloid fibrils, a small oligomeric form of alp synuclein, which may be analogous to the Abeta protofibi was observed prior to the appearance of fibrils. This speci or a related one, rather than the fibril itself, may be responsible for neuronal death.

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Accumulation of insoluble alpha-synuclein in dementia with Lewy bodies.

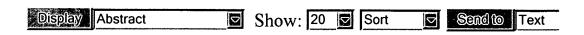
Campbell BC, Li QX, Culvenor JG, Jakala P, Cappai Beyreuther K, Masters CL, McLean CA.

Department of Pathology, The University of Melbourne, 3010, Australia.

The alpha-synuclein (alpha SN) protein is thought to play central role in the pathogenesis of neurodegenerative dise. where it aggregates to form intracellular inclusions. We have used Western blotting to examine the expression levels ar solubility of alpha SN in brain homogenates from dement with Lewy bodies (DLB), Parkinson's disease (PD), Alzheimer's disease (AD), and normal controls using sam from the parahippocampus/transentorhinal cortex. Compa to controls, DLB brains accumulate significantly greater amounts of sodium dodecyl sulfate (SDS)-soluble and SD insoluble alpha SN but levels of TBS-soluble alpha SN di not change. Levels of synaptophysin, a marker of synaptic integrity, were significantly lower in DLB cases than in normal aged controls regardless of whether concurrent changes of AD were present. This limbic synaptic dysfunction may contribute to cognitive impairment in DI Whether aggregated alpha SN is a cause or effect of the

disease process in DLB and PD remains to be determined the presence of aggregated alpha SN is consistent with a pathogenesis similar to that associated with aggregates of Abeta amyloid in AD. Copyright 2000 Academic Press.

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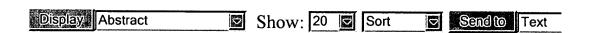
Beginning with the isolation of the fragment of alphasynuclein (alpha-syn) known as the non-Abeta componen amyloid plaques (NAC peptide) from Alzheimer's disease (AD) brains, alpha-syn has been increasingly implicated i the pathogenesis of neurodegenerative diseases, which no are classified as synucleinopathies. Indeed, unequivocal evidence linking abnormal alpha-syn to mechanisms of bi degeneration came from discoveries of missense mutation the alpha-syn gene pathogenic for familial Parkinson's dis (PD) in rare kindreds. Shortly thereafter, alpha-syn was shown to be a major component of Lewy bodies (LBs) an Lewy neurites in sporadic PD, dementia with LBs (DLB) the LB variant of AD. Also, studies of brains from patient with AD caused by genetic abnormalities demonstrated m alpha-syn positive LBs. Further, alpha-syn was implicated the formation of the glial (GCIs) and neuronal cytoplasmi inclusions of multiple system atrophy, and the LBs, GCIs neuraxonal spheroids of neurodegeneration with brain iro

accumulation type 1. Recently, two other members of the synuclein family, beta- and gamma-synuclein, have also be recognized to play a role in the pathogenesis of novel axo lesions in PD and DLB. Evidence for a role of alpha-syn i the formation of filamentous aggregates was reinforced by vitro studies showing aggregation and fibrillogenesis of mutant and wild type alpha-syn. Indeed, since the aggrega of brain proteins into presumptively toxic lesions is emerg as a common but poorly understood mechanistic theme in sporadic and hereditary neurodegenerative diseases. clarification of the mechanism of synuclein aggregation c augment efforts to develop novel and more effective thera for many neurodegenerative disorders. Copyright 2000 Wiley-Liss, Inc.

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Lewy body pathology in Alzheimer's disease.

Kotzbauer PT, Trojanowsk JQ, Lee VM.

Center for Neurodegenerative Disease Research, Universi of Pennsylvania School of Medicine, Philadelphia, USA.

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Lewy bodies, the characteristic pathological lesion of substantia nigra neurons in Parkinson's disease (PD), are frequently observed to accompany the amyloid plague and neurofibrillary tangle pathology of Alzheimer's disease (A However the typical anatomic distribution of Lewy bodies AD is distinct from PD. The most common site of occurre is the amygdala, where Lewy bodies are observed in approximately 60% of both sporadic and familial AD. Oth common sites of occurrence include the periamygdaloid a entorhinal cortex, while neocortical and brainstem areas develop Lewy bodies in a lower percentage of cases. In contrast, dementia with Lewy bodies (DLB), defined by widespread neocortical and brainstem Lewy bodies but frequently accompanied by variable levels of AD-type pathology, represents the other end of a spectrum of pathology associated with dementia. The observation of L bodies in familial AD cases suggests that like neurofibrill tangles, the formation of Lewy bodies can be induced by 1 pathological state caused by Abeta-amyloid overproduction

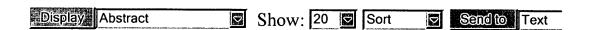
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The role of Lewy body formation in the dysfunction and degeneration of neurons remains unclear. The protein alpl synuclein appears to be an important structural componen Lewy bodies, an observation spurred by the discovery of point mutations in the alpha-synuclein gene linked to rare cases of autosomal dominant PD. Further investigation of alpha-synuclein and its relationship to pathological condit promoting Lewy body formation in AD, PD, and DLB ma yield further insight into pathogenesis of these diseases.

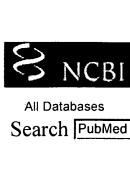
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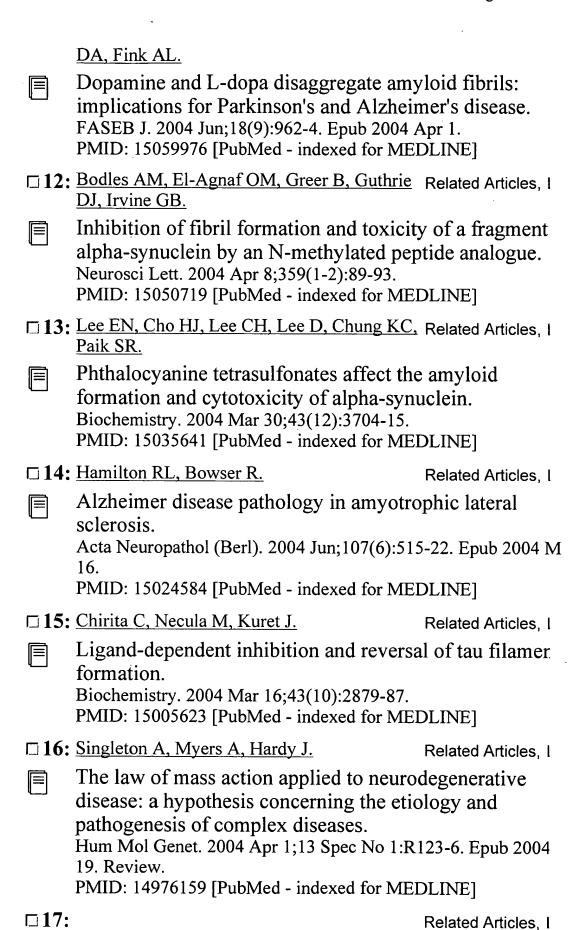


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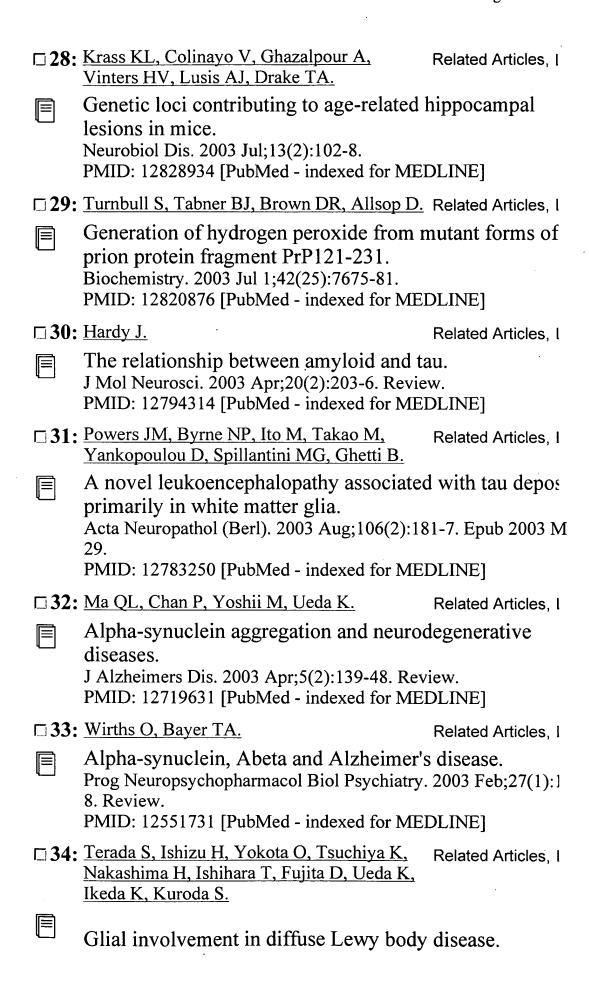
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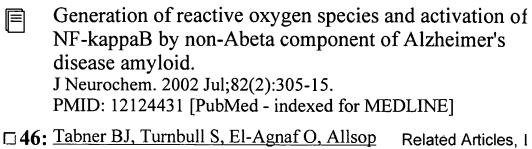
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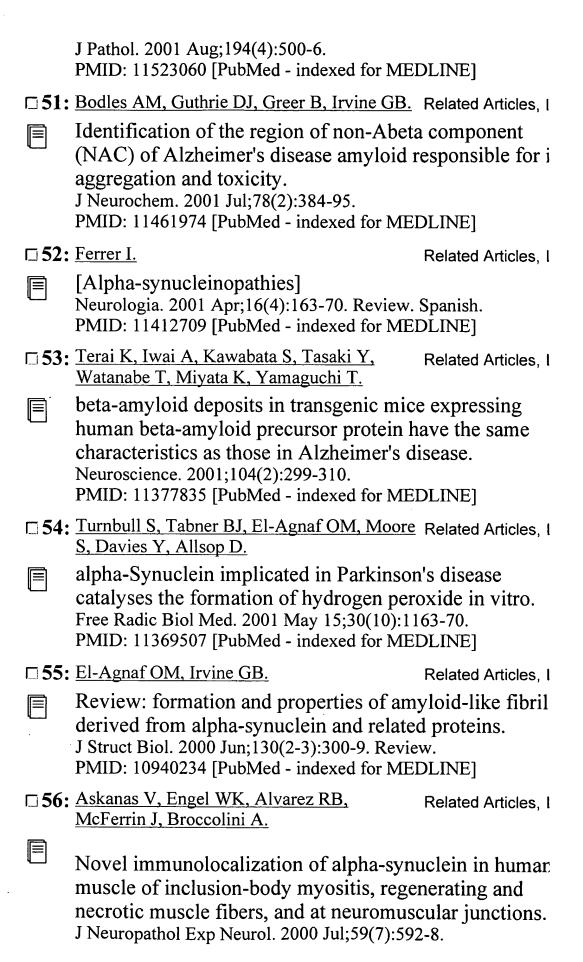
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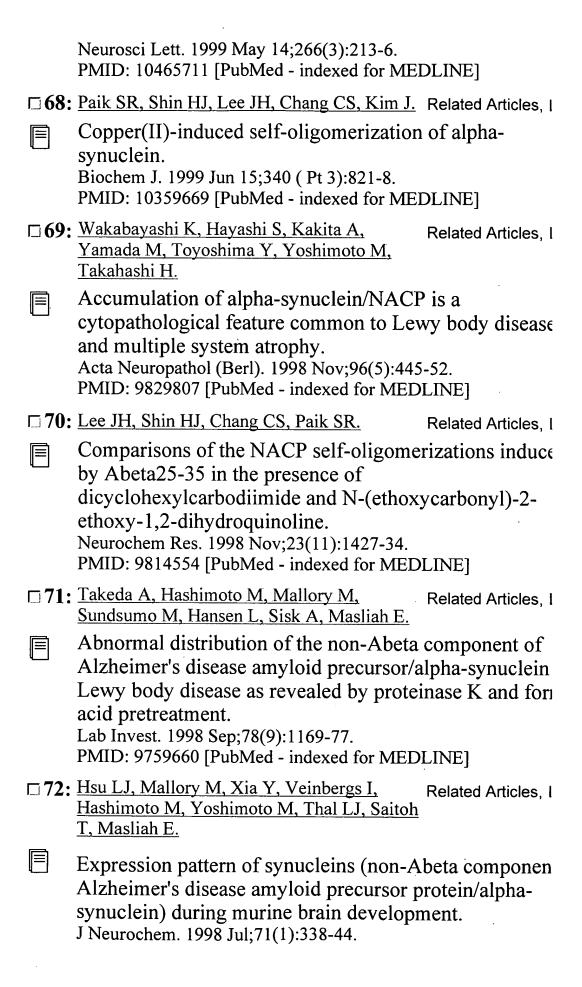
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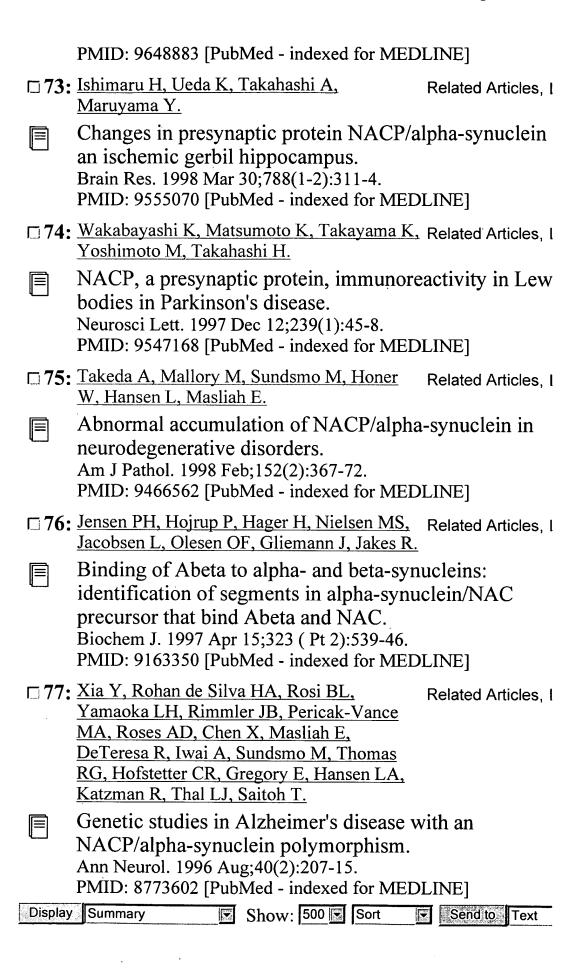
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Binding of Abeta to alpha- and beta-synucleins: identification of segments in alpha-synuclein/NA

precursor that bind Abeta and NAC.

Jensen PH, Hojrup P, Hager H, Nielsen MS, Jacobsen Olesen OF, Gliemann J, Jakes R.

Department of Medical Biochemistry, University of Aarh Ole Worms Alle, Building 170, DK-8000 Aarhus C, Denmark.

NAC, a 35-residue peptide derived from the neuronal proalpha-synuclein/NAC precursor, is tightly associated with Abeta fibrils in Alzheimer's disease amyloid, and alphasynuclein has recently been shown to bind Abeta in vitro. have studied the interaction between Abeta and synuclein aiming at determining segments in alpha-synuclein that ca account for the binding, as well as identifying a possible interaction between Abeta and the beta-type synuclein. W report that Abeta binds to native and recombinant alphasynuclein, and to beta-synuclein in an SDS-sensitive interaction (IC50 approx. 20 microM), as determined by chemical cross-linking and solid-phase binding assays. all Synuclein and beta-synuclein were found to stimulate Abo aggregation in vitro to the same extent. The synucleins als

displayed Abeta-inhibitable binding of NAC and they we capable of forming dimers. Using proteolytic fragmentation of alpha-synuclein and cross-linking to 125I-Abeta, we identified two consecutive binding domains (residues 1-5) and 57-97) by Edman degradation and mass spectrometric analysis, and a synthetic peptide comprising residues 32-5 possessed Abeta-binding activity. To test further the possi significance in pathology, alpha-synuclein was biotinylate and shown to bind specifically to amyloid plagues in a bra with Alzheimer's disease. It is proposed that the multiple Abeta-binding sites in alpha-synuclein are involved in the development of amyloid plaques.

PMID: 9163350 [PubMed - indexed for MEDLINE]

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Toxicity of non-abeta component of Alzheimer's Help | FAQ disease amyloid, and N-terminal fragments there correlates to formation of beta-sheet structure a fibrils.

> Bodles AM, Guthrie DJ, Harriott P, Campbell P, Irvin GB.

Centre for Peptide and Protein Engineering, School of Biology and Biochemistry, The Queen's University of Belfast, Northern Ireland.

The non-Abeta component of Alzheimer's disease amyloi-(NAC) and its precursor alpha-synuclein have been linked amyloidogenesis in Alzheimer's disease (AD), Parkinson's disease (PD) and dementia with Lewy bodies (DLB). Previously we have shown that NAC forms beta-sheet structures and fibrils [El-Agnaf, O.M.A., Bodles, A.M., Guthrie, D.J.S., Harriott, P. & Irvine, G.B. (1998) Eur. J. Biochem. 258, 157-163]. As a measure of their neurotoxic potential we have examined the ability of fresh and aged NAC and fragments thereof to inhibit the reduction of the redox dye 3-(4, 5-dimethylthiazol-2-yl)-2,5 diphenyltetrazolium bromide by rat pheochromocytoma P

thereof display varying degrees of toxicity. On immediate dissolution and after an incubation period for 3 days at 37 degrees C the full-length peptide and fragments NAC(3-1 and NAC(1-18) scrambled sequence [NAC(1-18 s)] were toxic, whereas fragments NAC(1-13) and NAC(6-14) wer not. CD indicates that NAC(3-18) and NAC(1-18 s) exhit beta-sheet secondary structure in aqueous solution, where NAC(1-13) and NAC(6-14) do not. NAC(3-18) aggregate as indicated by concentration of peptide remaining in solu after 3 days measured by an HPLC assay, and forms fibril as determined by electron microscopy. However, although some fibrils were detected for NAC(1-18 s) it does not co. out of solution to a significant degree. Fragments NAC(1and NAC(6-14) form few fibrils and remain in solution. These findings indicate that the ability of the central regio NAC to form beta-sheet secondary structures is important determining the toxicity of the peptide. This contrasts with what has been reported previously for most Abeta peptide their toxicity appears to require the peptide to have forme fibrillary aggregates as well as displaying beta-sheet. The: results suggest that an intermediate, which exhibits beta-s. structure, may be responsible for the toxic properties of N and provides further evidence for the role of NAC in the pathogenesis of AD, PD and DLB.

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Alpha-synuclein is a neuronal protein originally identified Alzheimer's disease (AD) amyloid plaques in 1993 and named non-Abeta component precursor (NACP) [92]. Lat the discovery of two missense mutations (G88C and G209 which resulted in Ala30Pro (A30P) and Ala53Thr (A53T) substitutions, of the alpha-synuclein gene in certain autosomal-dominant early onset familial Parkinson's disea (PD) has greatly promoted the understanding of the role o alpha-synuclein in the pathogenesis of neurodegenerative diseases, such as PD, dementia with Lewy bodies (DLB): multiple system atrophy (MSA) [5,6,51,75]. At present, it widely accepted that alpha-synuclein may play a central rein several neurodegenerative disorders because of the presence of insoluble alpha-synuclein as the major fibrilla component of inclusion bodies. From the cloning of the human alpha-synuclein cDNA in 1993 to the present, alph synuclein has been carefully documented in many aspects this article, we review the progress of studies on alphasynuclein and its role in alpha-synuclein-related neurodegenerative diseases.

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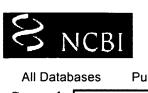
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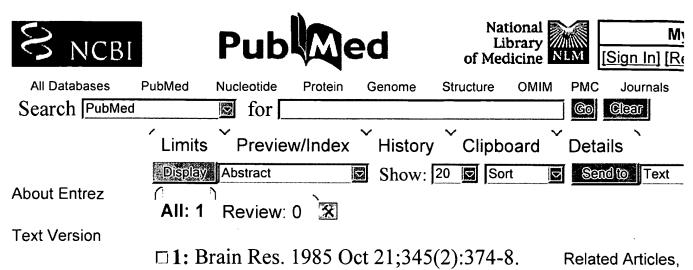
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Monoclonal antibodies which immunocytochemically lab Lewy bodies on sections of substantia nigra from subjects with Parkinson's disease were produced by immunization mice with substantia nigra and locus coeruleus containing Lewy bodies from parkinsonian subjects post-mortem. Te of specificity indicate that the antibodies do not recognize same antigen. One of the antibodies (G7) immunocytochemically labels only Lewy bodies, the othe (G9) also faintly labels the cell bodies of nigral dopamine neurons and cerebellar Purkinje cells in both normal and parkinsonian brains. Absorption experiments show, howe that the G7 antigen is present in normal substantia nigra a the G9 antigen in normal substantia nigra and Purkinje ce Neither of the antibodies seems to be directed against neurofilament protein. Immunoblots after two-directional electrophoresis indicate that antibody G7 labels a protein an iso-electric point around 5.6 and a mol. wt. of approximately 40 kdalton, whereas the protein labeled by antibody G9 has an iso-electric point of near 8 and a mol. above 70 kdalton.



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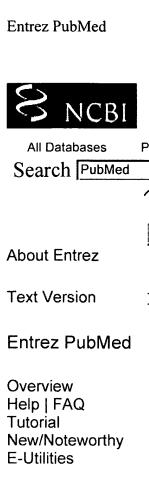
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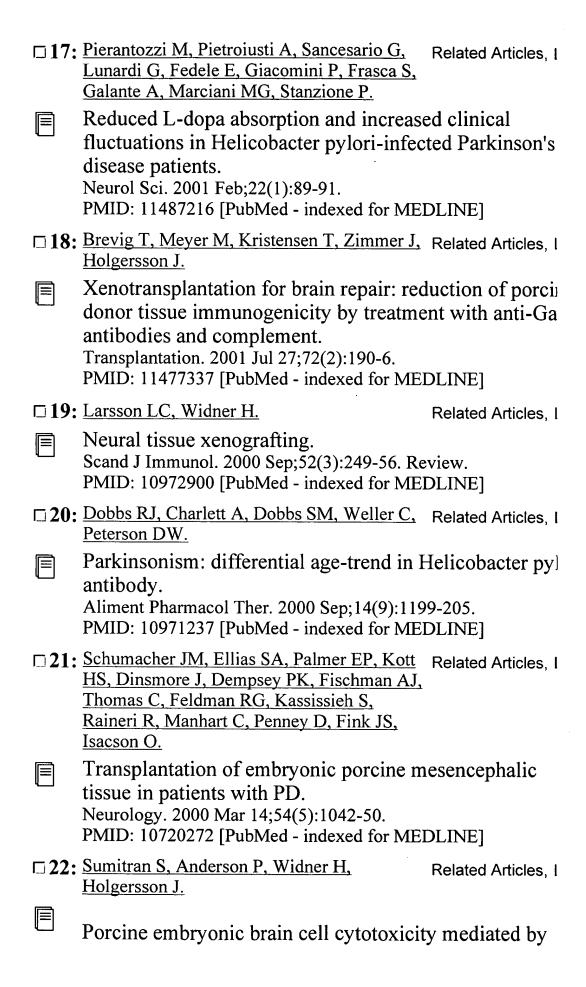
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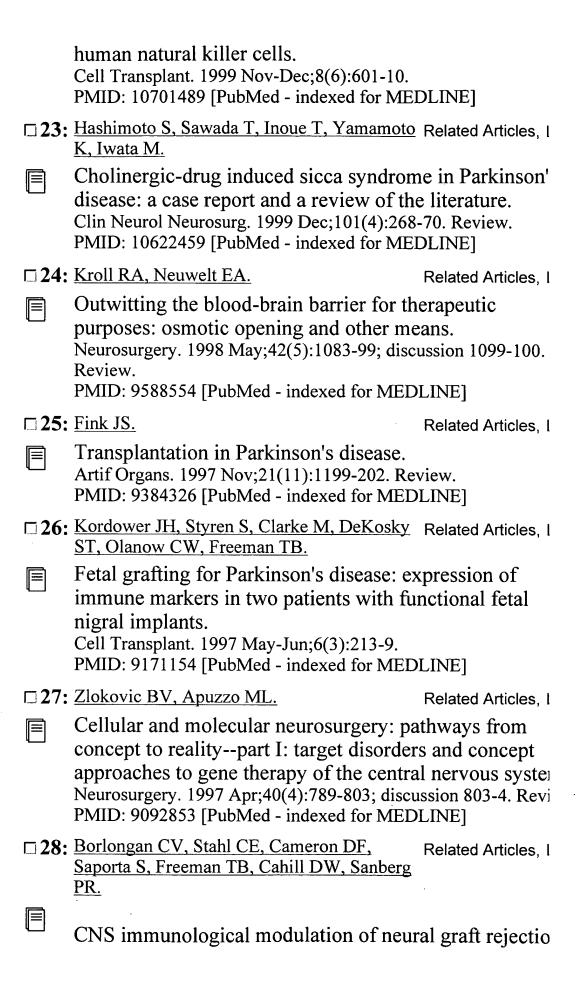
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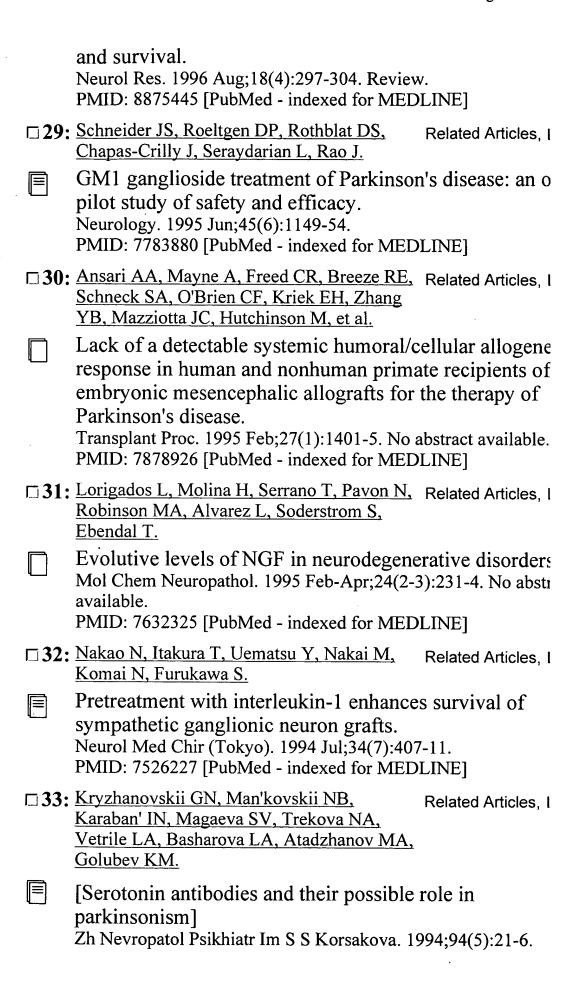
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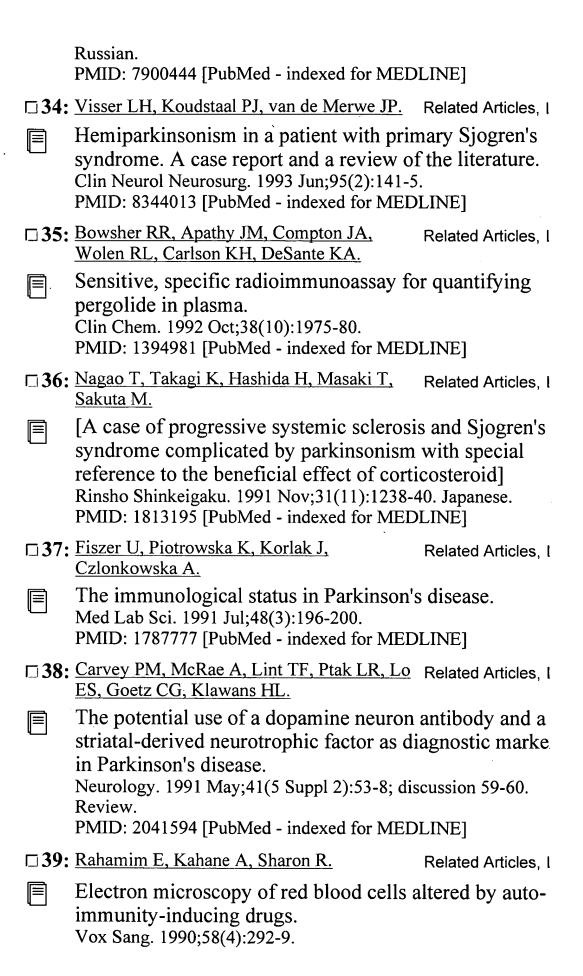
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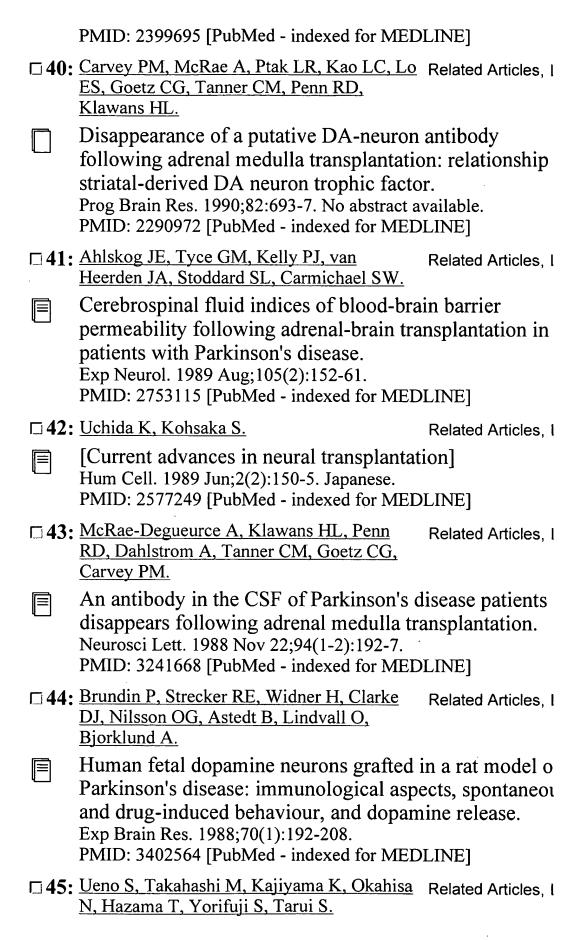
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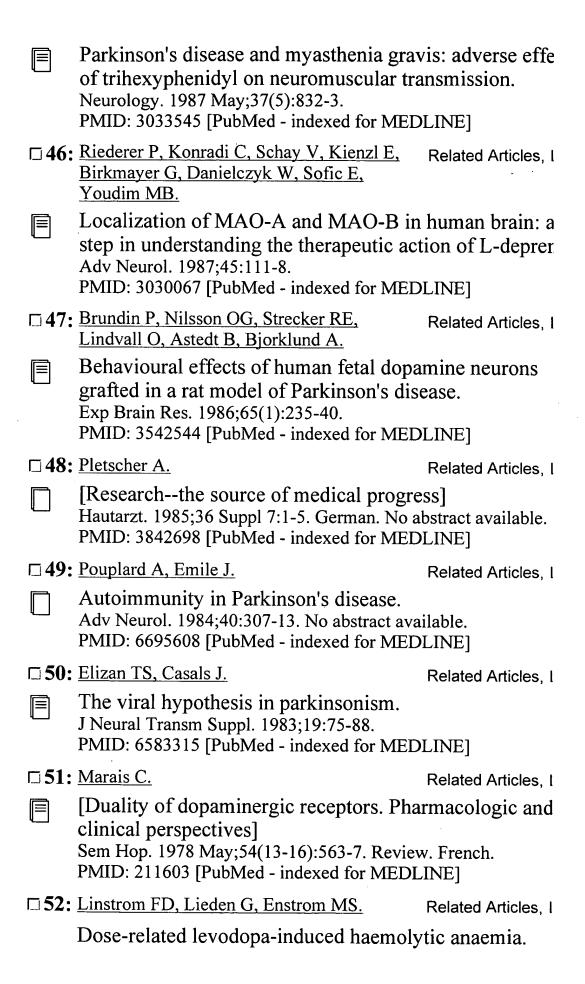












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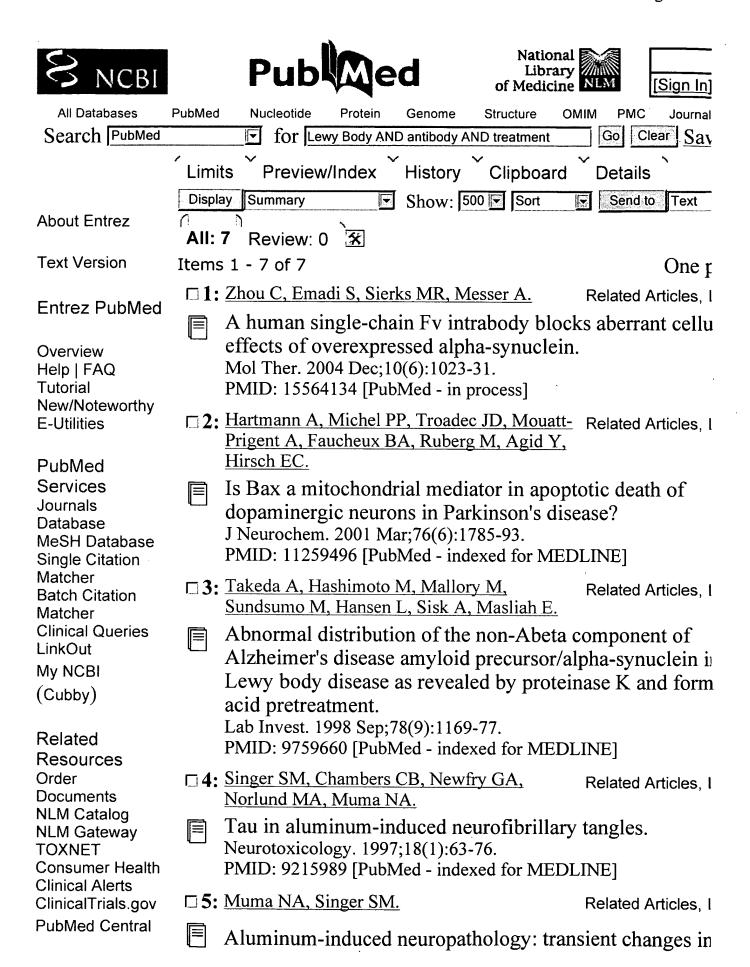
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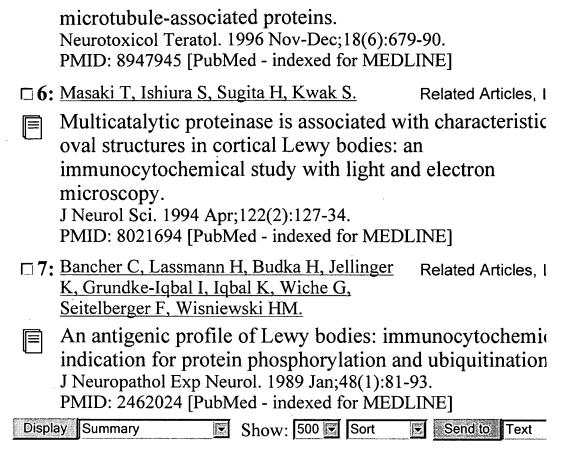
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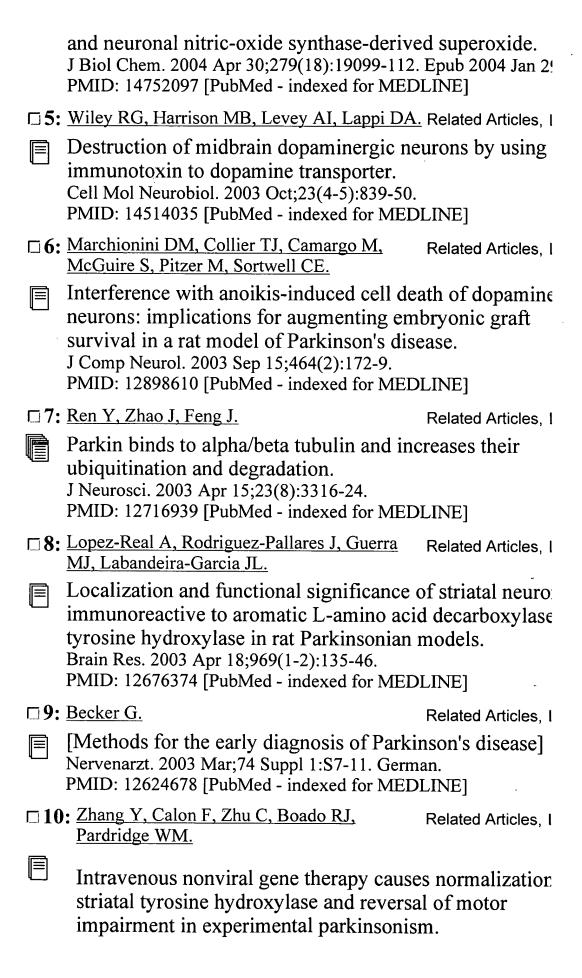




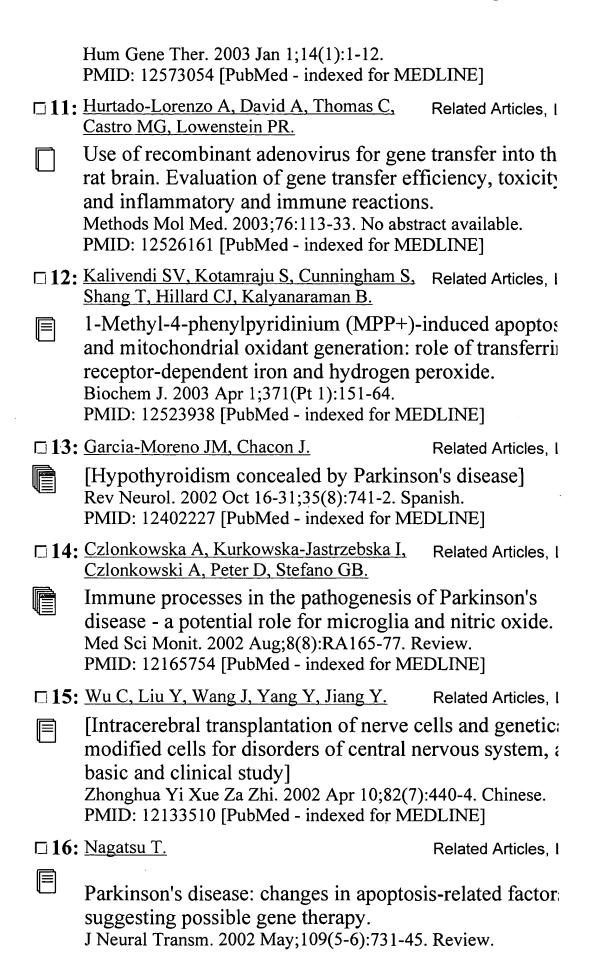


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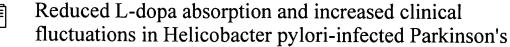
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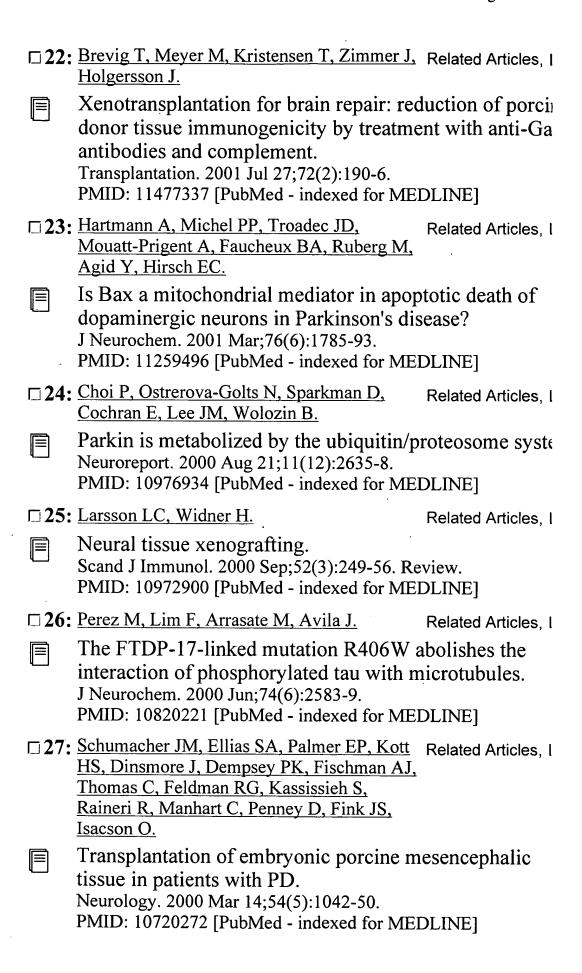
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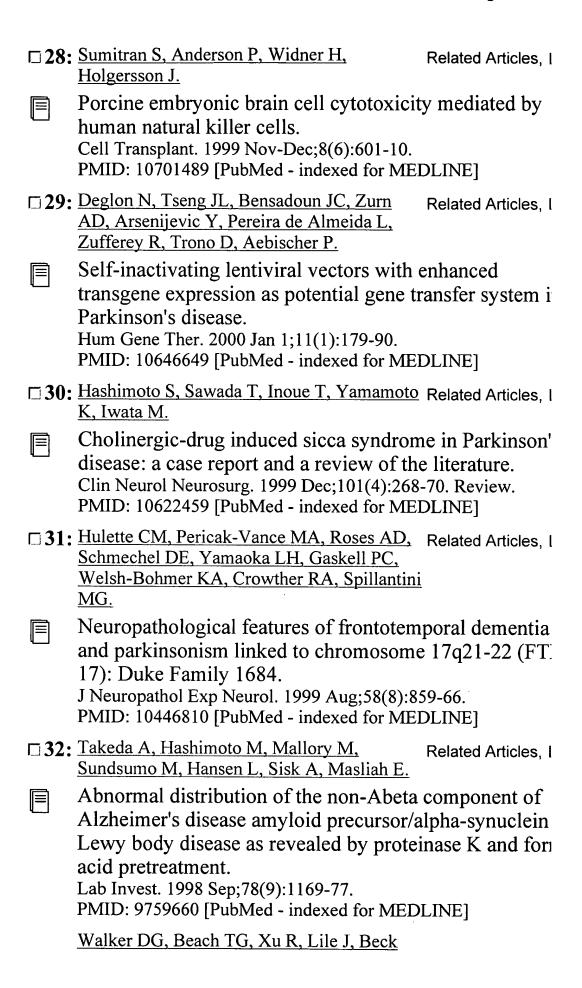


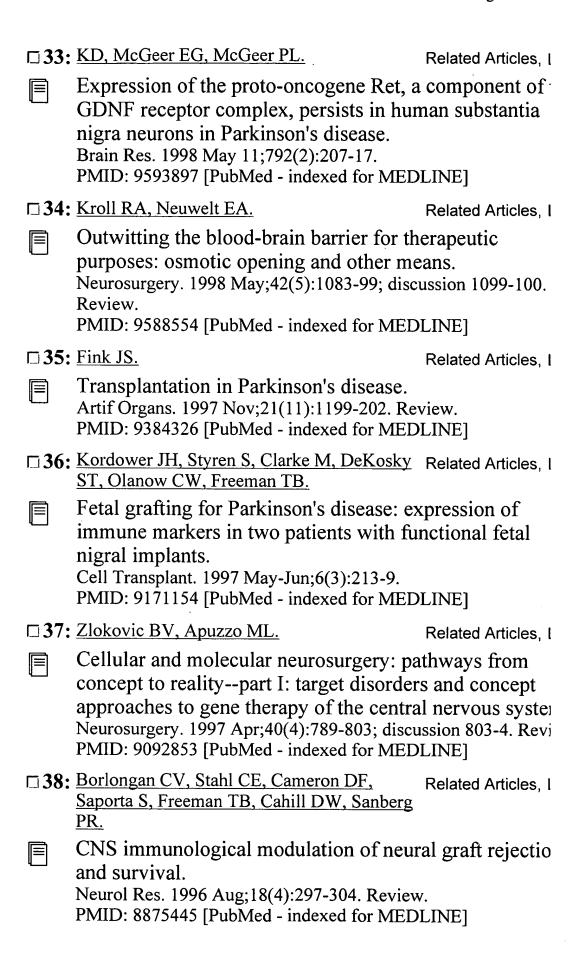
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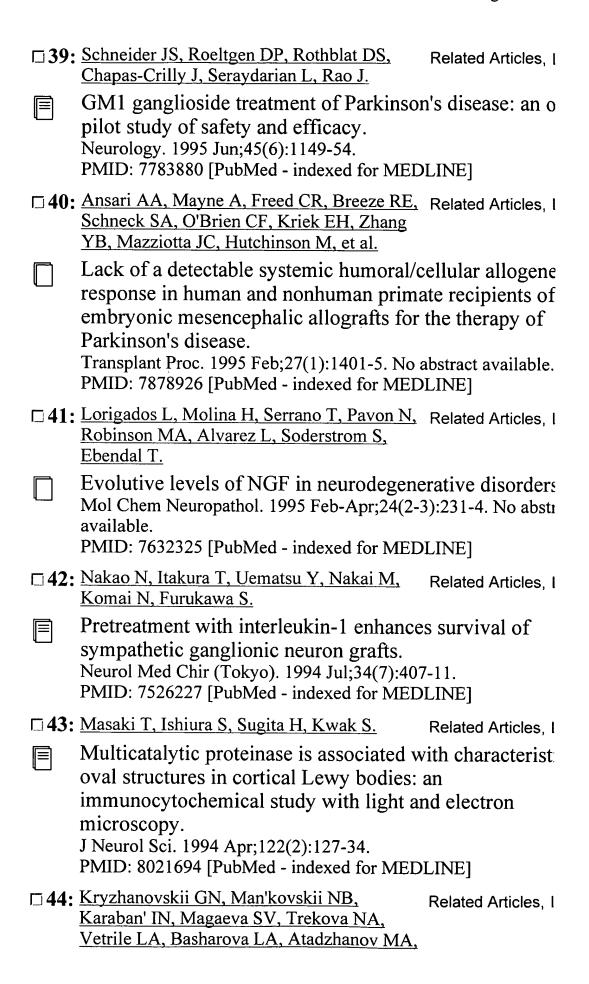
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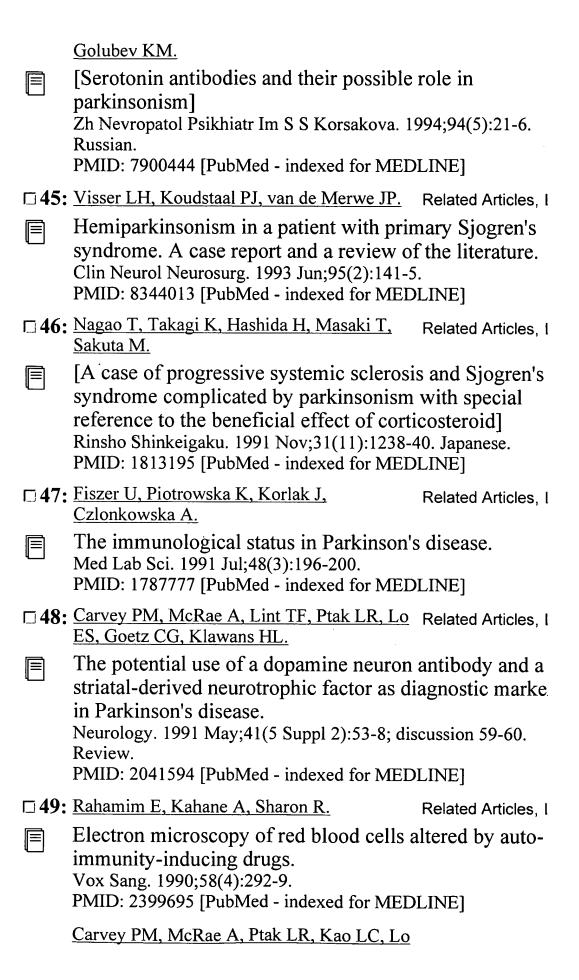
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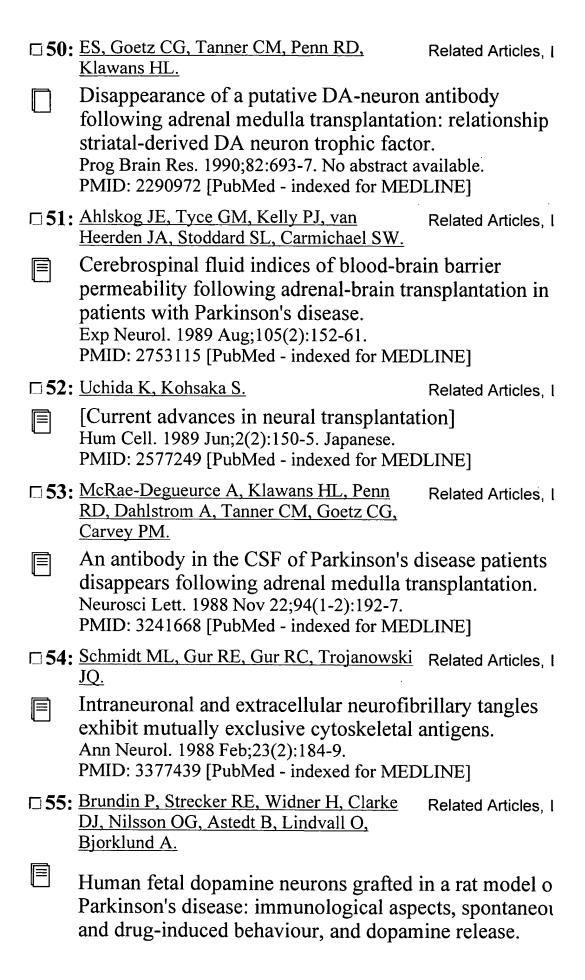


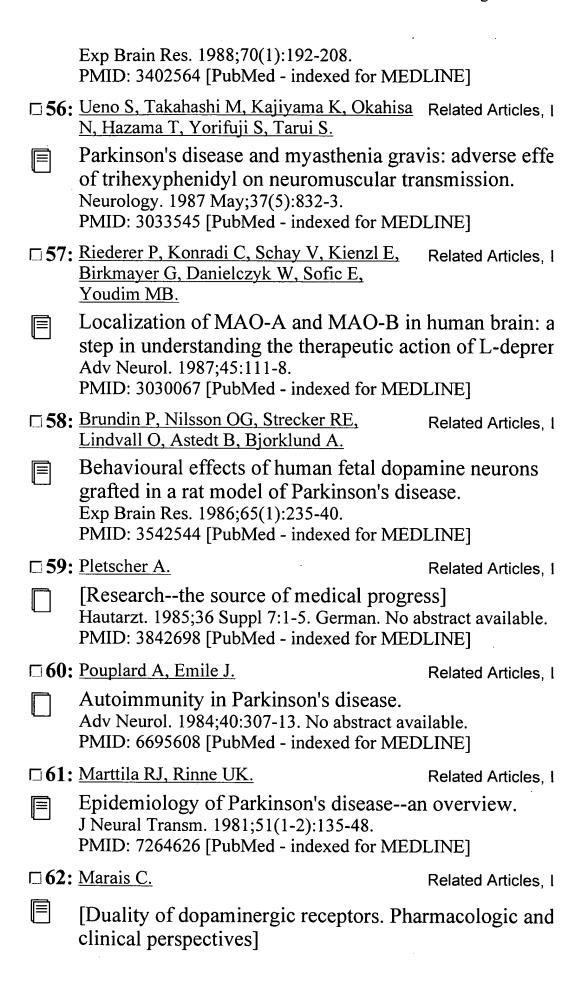












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	Endocrine function and glucose metabolism with Parkinson's disease and their altern J Clin Endocrinol Metab. 1971 Nov;33(5):829 available. PMID: 5125386 [PubMed - indexed for MED]	ation by L-Dop 9-37. No abstract			
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□ 69:	Nagay B.	Related Articles, I			
	[Dupuytren's contracture] Wiad Lek. 1970 Nov 15;23(22):1979-83. Rev abstract available. PMID: 4922139 [PubMed - indexed for MED]				

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4	Autoimmunity in patients treated with JAMA. 1969 Feb 17;207(7):1353-4. No abst PMID: 5304532 [PubMed - indexed for ME	tract available.
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NEWS 8 DEC 15 MEDLINE update schedule for December 2004
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NEWS
     10 DEC 17
                COMPUAB reloaded; updating to resume; current-awareness
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NEWS
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     12 DEC 17
                CERAB reloaded; updating to resume; current-awareness
NEWS
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                THREE NEW FIELDS ADDED TO IFIPAT/IFIUDB/IFICDB
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     13 DEC 17
     14 DEC 30 EPFULL: New patent full text database to be available on STN
NEWS
     15 DEC 30 CAPLUS - PATENT COVERAGE EXPANDED
NEWS
     16 JAN 03 No connect-hour charges in EPFULL during January and
NEWS
                February 2005
                CA/CAPLUS - Russian Agency for Patents and Trademarks
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     17 FEB 25
                 (ROSPATENT) added to list of core patent offices covered
NEWS
     18 FEB 10
                STN Patent Forums to be held in March 2005
NEWS
     19 FEB 16
                STN User Update to be held in conjunction with the 229th ACS
                National Meeting on March 13, 2005
NEWS
     20 FEB 28
                PATDPAFULL - New display fields provide for legal status
                data from INPADOC
NEWS 21 FEB 28 BABS - Current-awareness alerts (SDIs) available
NEWS 22 FEB 28 MEDLINE/LMEDLINE reloaded
NEWS 23 MAR 02 GBFULL: New full-text patent database on STN
NEWS 24 MAR 03 REGISTRY/ZREGISTRY - Sequence annotations enhanced
NEWS 25 MAR 03 MEDLINE file segment of TOXCENTER reloaded
NEWS 26 MAR 22 KOREAPAT now updated monthly; patent information enhanced
NEWS 27 MAR 22 Original IDE display format returns to REGISTRY/ZREGISTRY
NEWS 28 MAR 22
                PATDPASPC - New patent database available
NEWS 29 MAR 22 REGISTRY/ZREGISTRY enhanced with experimental property tags
NEWS EXPRESS
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NEWS EXPRESS JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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PT

AΤ

WO 2002021141 14 Mar 2002

WO 2000-US27632 6 Sep 2000

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PRAI
     US 2000-255033 12 Dec 2000
DТ
      Patent
LA
      English
O.S.
      WPI: 2002-351803 [38]
L4
     ANSWER 3 OF 21 CAPLUS COPYRIGHT 2005 ACS on STN
AN
     2004:412731 CAPLUS
DN
     140:417964
TI
     Prevention and treatment of synucleinopathic disease by administering
     agents that induce a beneficial immunogenic response against
       ***bodies***
IN
     Schenk, Dale B.; Masliah, Eliezer
PA
     Elan Pharmaceuticals, Inc., USA; The Regents of the University of
     California
SO
     PCT Int. Appl., 78 pp.
     CODEN: PIXXD2
DT
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LA
     English
FAN.CNT 9
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                        KIND
                                          APPLICATION NO.
                                DATE
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     WO 2004041067
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PRAI US 2002-423012P
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T.4
     ANSWER 4 OF 21 IFIPAT COPYRIGHT 2005 IFI on STN
AN
      10243450 IFIPAT; IFIUDB; IFICDB
      NOVEL METHOD FOR DOWN-REGULATION OF AMYLOID; ADMINISTERING AN
TI
      AMYLOIDOGENIC POLYPEPTIDE OR SUBSEQUENCE THEREOF TO INDUCES PRODUCTION OF
      ANTIBODIES AGAINST THE AMYLOIDOGENIC POLYPEPTIDE; TREATMENT OF
      ALZHEIMER'S DISEASE
IN
      Jensen Martin Roland (DK); Nielsen Klaus Gregorius (DK); Rasmussen Peter
      Birk (DK)
PΑ
      Unassigned Or Assigned To Individual (68000)
рT
      US 2002187157 A1 20021212
ΑI
      US 2001-785215
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      PA 2000-265
                          20000221
      US 2000-186295P
                          20000301 (Provisional)
      US 2002187157
FT
                          20021212
DT
      Utility; Patent Application - First Publication
      00001000
FS
      CHEMICAL
      APPLICATION
CLMN
      58
GI
       1 Figure(s).
     FIG. 1: Schematic depiction of Autovac variants derived from the amyloid
      precursor protein with the purpose of generating antibody responses
      against the A beta protein A beta-43 (or C100). The APP is shown
      schematically at the top of the figure and the remaining schematic
      constructs show that the model epitopes P2 and P30 are substituted or
      inserted into various truncations of APP. In the figure, the black
      pattern indicates the APP signal sequence, two-way cross-hatching is the
      extracellular part of APP, dark vertical hatching is the transmembrane
      domain of APP, light vertical hatching is the intracellular domain of
      APP, coarse cross-hatching indicates the P30 epitope, and fine
      cross-hatching indicates the P2 epitope. The full line box indicates A
      beta-42/43 and the fullline box and the dotted line box together indicate
      C-100. " ***Abeta*** " denotes A beta .
     ANSWER 5 OF 21 USPATFULL on STN
L4
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Therapeutic formulations for the treatment of beta-amyloid related

AN

ΤI

2005:36976 USPATFULL

diseases

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IN
       Gervais, Francine, Ile Bizard, CANADA
       Bellini, Francesco, Mount Royal, CANADA
PΙ
       US 2005031651
                           Α1
                                20050210
AΤ
       US 2004-871537
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                                20040618 (10)
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       Continuation-in-part of Ser. No. US 2003-746138, filed on 24 Dec 2003,
       PENDING
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       WO 2003-CA2011
                            20031224
       US 2002-436379P
                            20021224 (60)
       US 2003-482214P
                            20030623 (60)
       US 2003-480984P
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       ICM: A61K009-00
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
     ANSWER 6 OF 21 USPATFULL on STN
ΑN
       2005:10897 USPATFULL
ΤI
       Genes and polymorphisms on chromosome 10 associated with Alzheimer's
       disease and other neurodegenerative diseases
IN
       Becker, Kenneth David, San Diego, CA, UNITED STATES
       Velicelebi, Gonul, San Diego, CA, UNITED STATES
       Elliott, Kathryn J., San Diego, CA, UNITED STATES
       Wang, Xin, San Diego, CA, UNITED STATES
       Tanzi, Rudolph E., Hull, MA, UNITED STATES
       Bertram, Lars, Boston, MA, UNITED STATES
       Saunders, Aleister J., Philadelphia, PA, UNITED STATES
       Mullin, Kristina M., Weymouth, MA, UNITED STATES
       Sampson, Andrew Joseph, Oakwood, OH, UNITED STATES
PΙ
       US 2005009031
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       US 2003-600009
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       US 2001-339525P
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CAS INDEXING IS AVAILABLE FOR THIS PATENT.
L4
     ANSWER 7 OF 21 USPATFULL on STN
       2004:334837 USPATFULL
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       Method for the prediction, diagnosis and differential diagnosis of
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       Alzheimer's disease
IN
       Vanderstichele, Hugo, Gent, BELGIUM
       Vanmechelen, Eugeen, Nażareth-Eke, BELGIUM
       De Meyer, Geert, Gent, BELGIUM
       Blennow, Kaj, Goteborg, SWEDEN
       Kostanjevecki, Vesna, Sint-Denijs-Westrem, BELGIUM
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AN
       2004:327335 USPATFULL
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       Methods of engineering spatially conserved motifs in polypeptides
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       Chan, John, Raleigh, NC, UNITED STATES
       Zhang, Shengsheng, Framingham, MA, UNITED STATES
       Baynes, Brian, Somerville, MA, UNITED STATES
PA
       Compound Therapeutics, Inc., Waltham, MA (U.S. corporation)
PΙ
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       ICS: G06F019-00; G01N033-48; G01N033-50
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T.4
     ANSWER 9 OF 21 USPATFULL on STN
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       Peptides and methods of screening immunogenic peptide vaccines against
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       Chain, Daniel G., Jerusalem, ISRAEL
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CAS INDEXING IS AVAILABLE FOR THIS PATENT.
T.4
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       2004:107249 USPATFULL
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IN
       Afeyan, Noubar B., Lexington, MA, UNITED STATES
       Lee, Frank D., Chestnut Hill, MA, UNITED STATES
       Wong, Gordon G., Brookline, MA, UNITED STATES
       Das Gupta, Ruchira, Auburndale, MA, UNITED STATES
       Baynes, Brian, Somerville, MA, UNITED STATES
PΙ
       US 2004081648
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AΤ
       US 2003-650592
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PRAI
       US 2002-406517P
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       US 2002-423754P
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       US 2002-430001P
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DT
       Utility
FS
       APPLICATION
LN.CNT 8325
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       ICM: A61K038-48
       ICS: C12N009-64
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
     ANSWER 11 OF 21 USPATFULL on STN
L4
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       2004:107248 USPATFULL
TI
       Adzymes and uses thereof
IN
       Afeyan, Noubar B., Lexington, MA, UNITED STATES
       Lee, Frank D., Chestnut Hill, MA, UNITED STATES
       Wong, Gordon G., Brookline, MA, UNITED STATES
       DasGupta, Ruchira, Auburndale, MA, UNITED STATES
       Baynes, Brian, Somerville, MA, UNITED STATES
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       NCLS: 435/069.700; 435/226.000
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       ICS: C12N009-64; C12P021-04
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
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L4
       2004:51633 USPATFULL
AN
       Amine 1,2- and 1,3-diol compounds
TI
       Romero, Arthur G., Kalamazoo, MI, UNITED STATES
IN
       Schostarez, Heinrich J., Portage, MI, UNITED STATES
       Roels, Christina M., Battle Creek, MI, UNITED STATES
       US 2004039064
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       NCLS: 564/355.000
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       ICM: A61K031-137
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
L4
     ANSWER 13 OF 21 USPATFULL on STN
AN
       2004:44501 USPATFULL
ΤI
       Proteins and nucleic acids encoding same
IN
       Tchernev, Velizar T., Branford, CT, UNITED STATES
       Spytek, Kimberly A., New Haven, CT, UNITED STATES
       Zerhusen, Bryan D., Branford, CT, UNITED STATES
       Patturajan, Meera, Branford, CT, UNITED STATES
       Shimkets, Richard A., West Haven, CT, UNITED STATES
       Li, Li, Branford, CT, UNITED STATES
       Gangolli, Esha A., Madison, CT, UNITED STATES
       Padigaru, Muralidhara, Branford, CT, UNITED STATES
       Anderson, David W., Branford, CT, UNITED STATES
       Rastelli, Luca, Guilford, CT, UNITED STATES
       Miller, Charles E., Hill Drive, CT, UNITED STATES
       Gerlach, Valerie, Branford, CT, UNITED STATES
       Taupier, Raymond J., JR., East Haven, CT, UNITED STATES
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Gusev, Vladimir Y., UNITED STATES
       Colman, Steven D., Guilford, CT, UNITED STATES
       Wolenc, Adam Ryan, New Haven, CT, UNITED STATES
       Pena, Carol E. A., Guilford, CT, UNITED STATES
       Furtak, Katarzyna, Anosia, CT, UNITED STATES
       Grosse, William M., Bransford, CT, UNITED STATES
       Alsobrook, John P., II, Madison, CT, UNITED STATES
       Lepley, Denise M., Branford, CT, UNITED STATES
       Rieger, Daniel K., Branford, CT, UNITED STATES
       Burgess, Catherine E., Wethersfield, CT, UNITED STATES
PΙ
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       INCLS: 435/007.230; 435/069.300; 435/320.100; 435/325.000; 530/350.000;
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       ICM: C12Q001-68
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       C12N005-06; C07K014-47
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
L4
     ANSWER 14 OF 21 USPATFULL on STN
AN
       2004:18378 USPATFULL
TI
       Neurotoxic oligomers
IN
       Bush, Ashley, Somerville, MA, UNITED STATES
       Cherny, Robert, Victoria, AUSTRALIA
PΙ
       US 2004013680
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       US 2003-312437
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CAS INDEXING IS AVAILABLE FOR THIS PATENT.
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AN
       2003:325156 USPATFULL
       Aza hydroxylated ethyl amine compounds utility
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TN
       Schostarez, Heinrich, Portage, MI, UNITED STATES
       Chrusciel, Robert Alan, Portage, MI, UNITED STATES
       Centko, Rebecca S., Portage, MI, UNITED STATES
PТ
       US 2003229138
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              562/561.000; 564/147.000; 564/347.000; 564/464.000; 558/445.000
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       ICM: A61K031-275
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CAS INDEXING IS AVAILABLE FOR THIS PATENT.
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T.4
AN
       2003:318636 USPATFULL
TI
       Genes and polymorphisms on chromosome 10 associated with Alzheimer's
       disease and other neurodegenerative diseases
IN
       Becker, Kenneth David, San Diego, CA, UNITED STATES
       Velicelebi, Gonul, San Diego, CA, UNITED STATES
       Ellliott, Kathryn J., San Diego, CA, UNITED STATES
       Wang, Xin, San Diego, CA, UNITED STATES
       Tanzi, Rudolph E., Hull, MA, UNITED STATES
       Bertram, Lars, Brighton, MA, UNITED STATES
       Saunders, Aleister J., Philadelphia, PA, UNITED STATES
       Mullin, Kristina M., south Boston, MA, UNITED STATES
       Sampson, Andrew Joseph, Dayton, OH, UNITED STATES
       The General Hospital Corporation (U.S. corporation)
PA
ΡI
       US 2003224380
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ΑI
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DT
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LN.CNT 13662
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IC
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CAS INDEXING IS AVAILABLE FOR THIS PATENT.
     ANSWER 17 OF 21 USPATFULL on STN
L4
AN
       2003:225306 USPATFULL
ΤI
       Novel method for down-regulation of amyloid
IN
       Rasmussen, Peter Birk, Horsholm, DENMARK
       Jensen, Martin Roland, Horsholm, DENMARK
       Nielsen, Klaus Gregorius, Horsholm, DENMARK
       Koefoed, Peter, Horsholm, DENMARK
       Degan, Florence Dal, Horsholm, DENMARK
PΙ
       US 2003157117
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ΑI
       US 2002-223809
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       DK 2001-1231
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       US 2001-337543P
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DT
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       APPLICATION
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IC
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       ICS: C12N009-64
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
     ANSWER 18 OF 21 USPATFULL on STN
L4
AN '
       2003:135733 USPATFULL
ΤI
       Transgenic animal model of neurodegenerative disorders
IN
       St. George-Hyslop, Peter H., Toronto, CANADA
       Fraser, Paul E., Toronto, CANADA
       Westaway, David, Etobicoke, CANADA
PΙ
       US 2003093822
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LN.CNT 1380
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L4
     ANSWER 19 OF 21 USPATFULL on STN
AN
       2003:126727 USPATFULL
TI
       Novel methods for down-regulation of amyloid
       Jensen, Martin Roland, Horsholm, DENMARK
IN
       Birk, Peter, Horsholm, DENMARK
       Nielsen, Klaus Gregorius, Horsholm, DENMARK
PΙ
       US 2003086938
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       US 2002-204362
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LN.CNT 3114
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IC
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       ICM: A61K039-00
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
     ANSWER 20 OF 21 USPATFULL on STN
L4
ΑN
       2002:191196 USPATFULL
ΤI
       Methods and compositions for stimulating CD 45 and thereby suppressing
       microglial activation associated with Alzheimer's disease
TN
       Tan, Jun, Tampa, FL, UNITED STATES
       Town, Terrence, Tampa, FL, UNITED STATES
       Mullan, Michael, Tampa, FL, UNITED STATES
PΙ
       US 2002102259
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       US 2001-985598
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IC
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       ICM: A61K039-395
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
     ANSWER 21 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
AN
     2002-130835 [17]
                        WPIDS
DNC
     C2002-040215
     Treatment and prevention of conditions characterized by pathological
     aggregation and accumulation of a specific protein associated with
     oxidative damage and tyrosine-cross-link formation..
DC
IN
     BUSH, A; CHERNY, R
     (GEHO) GEN HOSPITAL CORP; (PRAN-N) PRANA BIOTECHNOLOGY LTD; (BUSH-I) BUSH
PA
     A; (CHER-I) CHERNY R
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    WO 2002000245 A1 WO 2001-AU786 20010628; AU 2001068828 A AU 2001-68828
     20010628; EP 1296705 A1 EP 2001-947033 20010628, WO 2001-AU786 20010628;
     CN 1450908 A CN 2001-813312 20010628; US 2004013680 A1 WO 2001-AU786
     20010628, US 2003-312437 20030616; JP 2004501204 W WO 2001-AU786 20010628.
    JP 2002-505026 20010628
    AU 2001068828 A Based on WO 2002000245; EP 1296705 A1 Based on WO
     2002000245; JP 2004501204 W Based on WO 2002000245
PRAI US 2000-242177P
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     US 2003-312437
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         A61K039-395; A61P021-00; A61P025-02; A61P025-14; A61P025-16;
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A61P025-28; A61P027-12; C07K014-47; G01N033-53

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